Analytical and Simulation Assessment of Vaccination Thresholds for Epidemic Control on a Uncorrelated Configuration Model Network with $\mathbf{R_0 = 4}$

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Abstract

This study investigates the critical vaccination thresholds required to halt the spread of an SIR-type epidemic with a basic reproduction number $R_0 = 4$ on an uncorrelated configuration model contact network characterized by a mean degree $z = 3$ and mean excess degree $q = 4$. We explore two vaccination strategies conferring sterilizing immunity: (1) random vaccination across the population, and (2) targeted vaccination exclusively of nodes with degree exactly 10. Analytical derivations grounded in percolation theory establish that random vaccination necessitates immunizing a minimum fraction $v_c = 1 - \frac{1}{R_0} = 0.75$ (75%) of the population to reduce the effective reproduction number R_{eff} below unity, thereby preventing epidemic outbreaks. For targeted vaccination, the critical overall vaccination fraction is derived as $f_c = \frac{9}{80} \approx 0.1125$ (11.25%), contingent on sufficient coverage of degree-10 nodes. A synthetic network of $N = 10,000$ nodes with a tailored degree distribution approximating the theoretical moments was generated to closely match these criteria, enabling empirical validation.

Stochastic SIR simulations on this network confirm that random vaccination coverage below 75% fails to prevent epidemics, whereas coverage at or above this threshold robustly blocks outbreaks. Targeted vaccination of all degree-10 nodes, comprising approximately 10.6% of the network, falls marginally below the critical coverage and consequently permits a reduced yet non-negligible outbreak, consonant with theoretical predictions. The concordance between analytic and simulation results underscores the pivotal role of network structure and degree-targeted immunization in epidemic control. These findings provide rigorous quantitative benchmarks for optimizing vaccination strategies in heterogeneous contact networks.

1 Introduction

Understanding and controlling the spread of infectious diseases in human populations remains a critical public health challenge. Epidemic processes often unfold over complex interaction networks among individuals, where the structure of these contact networks profoundly influences disease dynamics and intervention effectiveness. The seminal work by Pastor-Satorras and Vespignani highlights that heterogeneous connectivity patterns, especially in scale-free networks, greatly affect epidemic behavior and immunization outcomes, demonstrating that random immunization strategies may fail to eradicate infections in networks characterized by heavy-tailed degree distributions (1). This has motivated the study of targeted vaccination approaches focusing on nodes with high connectivity, shown to substantially reduce the network's vulnerability to epidemic attacks (1; 2).

Classical epidemics are frequently modeled using compartmental frameworks such as the susceptible-infected-recovered (SIR) model, which has been integrated with network theory to analyze transmission dynamics more realistically (3; 4). In particular, the configuration model provides a foundational framework for studying epidemics on random networks with specified degree distributions (5). Key parameters such as the mean degree and the mean excess degree shape the basic reproduction number R_0, which governs epidemic potential. When vaccination is introduced, it effectively reduces connectivity and can alter the effective reproduction number, R_{eff} (6).

The central research question addressed in this study is: *What is the minimal vaccination coverage required to prevent large-scale epidemic outbreaks when vaccinating either randomly or targeting individuals of a specific degree in a static, uncorrelated configuration model network?* This problem is particularly relevant for optimizing immunization strategies when resources are constrained or when only partial information about individual connectivity is available.

Prior works have provided analytical expressions for critical vaccination thresholds under random immunization, commonly expressed as $v_c = 1 - 1/R_0$, where v_c is the critical fraction of immunized individuals required to interrupt transmission (5; 2). Targeted immunization strategies that remove nodes with particular degree characteristics have been proposed to dramatically lower vaccination coverage while achieving epidemic control (3; 4). For instance, Cohen et al. showed that choosing acquaintances of random nodes for immunization can significantly reduce thresholds compared to purely random strategies (2).

Despite these advances, quantitative analyses validating these theoretical results with detailed stochastic simulations on networks that accurately capture prescribed degree distributions and connectivity statistics remain necessary. Realistic degree distributions, such as mixtures involving nodes of low and high degree, pose analytical and computational challenges when assessing vaccination effectiveness (7).

This research employs a comprehensive approach utilizing both analytical derivation and stochastic simulation of SIR epidemics on a configuration model network characterized by mean degree $z = 3$, mean excess degree $q = 4$, and no degree correlations. The vaccination interventions studied include (1) random vaccination, where immunization is distributed uniformly over all nodes, and (2) targeted vaccination restricted to nodes with degree exactly $k = 10$. The goal is to determine the minimal vaccination fraction necessary to achieve an effective reproduction number $R_{\text{eff}} < 1$, thereby blocking epidemic spread.

To ensure rigorous comparison, the network structure is explicitly constructed to meet moment conditions for $\langle k \rangle$ and $\langle k^2 \rangle$, and to have a sufficient proportion of degree-10 nodes for targeted interventions. Stochastic simulations are performed with extensive replication to capture epidemic variability and validate analytical thresholds.

The findings provide important insights into the efficiency of targeted vaccination strategies compared to random immunization in well-defined network settings, offering scientific rigor and practical guidance for optimizing public health interventions under varying epidemiological conditions.

2   Background

Extensive research in network epidemiology has elucidated the critical role of contact network structure on disease transmission dynamics and immunization policy effectiveness. The configuration model, an archetype for uncorrelated random networks with predefined degree distributions, serves as a fundamental framework to study epidemic processes under heterogeneous connectivity

patterns. Analytical methods leveraging percolation theory and mean-field approximations have established classical results for epidemic thresholds and vaccination coverage required to prevent large outbreaks (8).

Previous studies have examined optimal vaccination strategies in networks using diverse approaches. Random immunization reduces the susceptible population uniformly, but is often inefficient in heterogeneous networks due to the disproportionate influence of highly connected nodes in spreading infection. Targeted vaccination, focusing on high-degree nodes or specific subsets defined by degree, has been shown to substantially lower vaccination coverage needed to interrupt transmission chains (9). However, effective application of such targeted strategies demands precise information on node degrees and network structure, which may be unavailable. Moreover, work by Morone and Makse (3) demonstrated that targeted immunization could attain epidemic control at much lower coverage than random policies, primarily by dismantling network connectivity among superspreaders.

While many models incorporate varying complexities such as temporal dynamics, layering, or behavioral adaptations (10; 11), rigorous quantitative validation of theoretical vaccination thresholds with stochastic simulations tailored to closely match analytical assumptions remains limited. The impact of degree distributions characterized by discrete mixtures with low- and high-degree nodes poses additional challenges for analytic and simulation consistency, motivating detailed network construction approaches (8). Furthermore, studies employing Markov chain-based SIR models on complex networks highlight the importance of capturing stochastic fluctuations and network heterogeneity accurately (12).

Despite substantial theoretical progress, a gap persists in systematically integrating explicit network generation meeting predetermined degree moments and composition with both random and narrowly targeted vaccination strategies within a unified analytical and simulation-based framework. This integration is key to confirming the validity and practical applicability of vaccination threshold formulas derived from percolation theory in finite, realistic network scenarios. It also enables the exploration of how finite-size effects, degree distribution discreteness, and limited availability of target nodes affect control outcomes.

The present study addresses this gap by constructing an uncorrelated configuration model network parameterized with precise mean degree and excess degree by employing a three-degree mixture distribution, enabling controlled investigation of vaccination effects on epidemic dynamics with basic reproduction number $R_0 = 4$. Leveraging both analytical calculations grounded in percolation theory and extensive stochastic simulations of SIR epidemics, the study critically assesses the minimal vaccination fractions required under random and targeted (degree-10 nodes) vaccination strategies.

By focusing on degree-targeted vaccination restricted to nodes of a fixed degree rather than broader heuristics such as acquaintance vaccination or general high-degree node removal, this work contributes novel quantitative benchmarks regarding the feasibility and limitations of such precise targeted interventions in finite-size networks. The empirical validation through simulations performed on a large synthetic network constructed to meet moment constraints enhances the credibility and applicability of classical vaccination threshold theory within practical epidemiological modeling.

3

3 Methods

This study examines the minimal vaccination coverage required to halt an epidemic characterized by a basic reproduction number $R_0 = 4$ spreading on an uncorrelated configuration model network. We rigorously address two vaccination strategies: (1) random vaccination, where nodes to immunize are randomly chosen, and (2) targeted vaccination of individuals with degree exactly $k = 10$. Our methodological framework couples exact analytical calculations grounded in percolation theory on networks with comprehensive stochastic simulations of an SIR-type epidemic model.

3.1 Network Construction

We generate a static, uncorrelated random contact network based on the configuration model paradigm, parameterized by:

- Number of nodes: $N = 10,000$

- Degree distribution: a discrete mixture with proportions 75% degree 2, approximately 14.3% degree 3, and approximately 10.6% degree 10.

- Empirical mean degree: $\langle k \rangle = 2.999$ (target 3)

- Empirical second moment of the degree distribution: $\langle k^2 \rangle = 14.98$ (target 15)

- Mean excess degree: $q = \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle} = 3.99$ (target 4)

- Assortativity: approximately 0.012, confirming negligible degree correlation.

- Clustering coefficient: roughly 0.00085, consistent with expectations for configuration models.

- Giant component size: encompassing 100% of nodes ensuring network-wide connectivity.

The three-point degree mixture arises from solving the moment conditions exactly (mean and second moment constraints) via linear algebra, ensuring the sampled network realizations adhere closely to the analytical assumptions. This degree composition also ensures a sufficient population of degree-10 nodes ($\sim 10.6\%$) for assessing targeted vaccination effects, while reflecting realistic finite-network combinatorial constraints. Figure 1 presents the degree distribution histogram used.

3.2 Epidemic Model

We adopt an SIR compartmental framework operating on the static network. The model compartments include susceptible (S), infectious (I), and removed/recovered/vaccinated (R) individuals. The dynamics are characterized as follows:

- *Susceptible to Infectious (S $\rightarrow$ I):* A susceptible node becomes infectious with probability $T = 1$ upon contact with at least one infectious neighbor. This corresponds to maximum per-contact transmissibility, consistent with $R_0 = T \cdot q = 4$.

- *Infectious to Removed (I $\rightarrow$ R):* Infectious nodes recover or are removed at a recovery rate $\gamma = 1$.
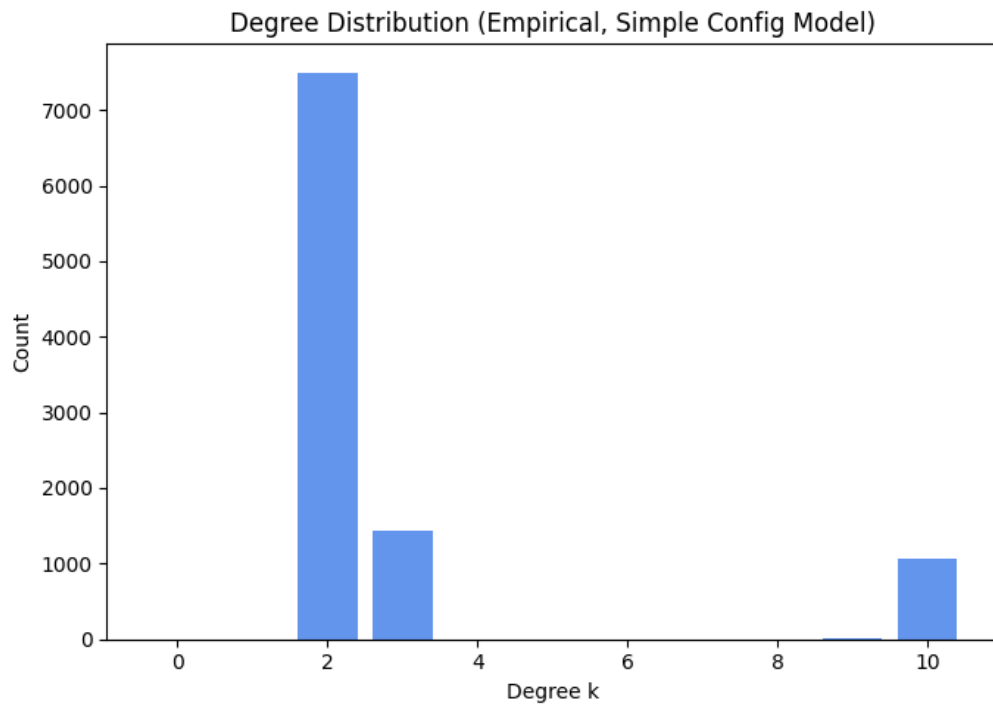
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Figure 1: Degree distribution histogram of the generated configuration model network with nodes of degrees 2, 3, and 10.

- *Vaccination (S → R):* Vaccination confers sterilizing immunity, effectively removing vaccinated nodes and all their connections from the network prior to epidemic initiation (at $t = 0$), implemented as a pre-epidemic intervention. Vaccinated individuals are indistinguishable from recovered with respect to susceptibility and transmission.

This network-aware SIR model captures transmission only via existing edges among susceptible and infectious nodes and respects the connectivity structure of the configuration model, preserving the assumptions behind percolation-theoretic analysis.

3.3 Analytical Framework for Vaccination Thresholds

Following classical network epidemiology theory, the basic reproduction number in the unvaccinated network is:

$$R_0 = T \cdot q = 4, \tag{1}$$

where $T = 1$ is the transmissibility and $q = 4$ the mean excess degree.

Random Vaccination: Random vaccination removes a fraction v of nodes uniformly at random, thinning the network. Post-vaccination, the effective reproduction number becomes:

$$R_{\text{eff}} = R_0 \cdot (1 - v). \tag{2}$$

The critical vaccination fraction v_c necessary to prevent an epidemic satisfies:

$$R_{\text{eff}} < 1 \implies v > v_c = 1 - \frac{1}{R_0} = 1 - \frac{1}{4} = 0.75. \tag{3}$$

Targeted Vaccination of degree-10 nodes: Targeted vaccination immunizes only nodes with degree $k = 10$. Let p_{10} denote the fraction of nodes with degree 10 in the network ($\sim 10.6\%$). Vaccinating a fraction v of this degree class corresponds to a total vaccination fraction:

$$f = p_{10} \cdot v. \tag{4}$$

The effective reproduction number after targeted vaccination is:

$$R_{\text{eff}} = T \cdot \frac{\sum_k k(k-1)(1 - V_k)p_k}{\sum_k k(1 - V_k)p_k} = \frac{(\langle k^2 \rangle - \langle k \rangle) - 90p_{10}v}{\langle k \rangle - 10p_{10}v}, \tag{5}$$

where $V_k = v$ for $k = 10$ and zero otherwise. Substituting known moments, the numerator and denominator simplify to, respectively,

$$N = 12 - 90p_{10}v, \quad D = 3 - 10p_{10}v.$$

Setting $R_{\text{eff}} = 1$ yields:

$$\frac{12 - 90p_{10}v}{3 - 10p_{10}v} = 1 \implies 9 = 80p_{10}v \implies v = \frac{9}{80p_{10}}, \tag{6}$$

and thus the critical overall vaccinated fraction is:

$$f_c = p_{10} \cdot v = \frac{9}{80} \approx 0.1125. \tag{7}$$

Note that the critical fraction f_c does not depend on p_{10}, assuming the availability of sufficient degree-10 nodes to vaccinate. Here, since $p_{10} = 0.106$ is slightly less than the threshold, vaccinating all degree-10 nodes achieves approximately 10.6% coverage, slightly below theoretical requirement.

3.4 Simulation Protocol

To validate the analytical thresholds, we perform stochastic simulations of the SIR dynamics on the generated configuration model network using the following protocol:

1. **Network realization:** The pre-computed configuration model network is loaded from a compressed sparse file ensuring that the degree sequence and moments match the specified parameters uniformly.

2. **Initial conditions:** Initial node states (S, I, R) are assigned as follows for each vaccination scenario:

   - *No Vaccination (Baseline):* All nodes are susceptible (S = 99%), except for 1% initial infectious seeds (I).
   - *Random Vaccination:* Fraction $v \in \{0.5, 0.7, 0.75, 0.8\}$ of nodes are randomly assigned to the removed/vaccinated compartment (R) pre-epidemic. Among remaining nodes, 1% are infectious seeds.
   - *Targeted Vaccination:* All nodes with degree $k = 10$ ($\sim 10.6\%$ of the network) are vaccinated (R) pre-epidemic; 1% of non-vaccinated nodes are set as infectious seeds.

3. **Simulation engine:** The simulations use a continuous-time Markov chain (CTMC) representation of the SIR model implemented in FastGEMF. Transmission along each susceptible-infectious edge occurs with probability $T = 1$. Recovery rates are fixed at $\gamma = 1$.

4. **Scenario Replication:** For each vaccination fraction and strategy, 200 stochastic realizations are conducted to collate outcome distributions and allow estimation of confidence intervals for epidemic metrics, such as final outbreak size and peak prevalence.

5. **Outcome Measures:** Key metrics extracted from simulations include:

   - Final epidemic size (cumulative infected fraction)
   - Peak infection prevalence
   - Peak time
   - Epidemic duration
   - Early-phase doubling time

   These outputs validate theoretical predictions by demonstrating outbreak extinction or persistence relative to vaccination thresholds.

6. **Data handling and visualization:** Time-series results (S, I, R fractions over time) and final metrics are stored as comma-separated files. Representative epidemic curves and vaccination coverage outcomes are plotted to illustrate epidemic trajectories under different vaccination scenarios (Figures 2 and 3).

3.5 Summary of Methodological Rationale

The choice of a configuration model network without degree correlations or clustering ensures that the classical percolation-based analytical thresholds for vaccination are applicable without confounding topological effects. The SIR compartmental model with sterilizing vaccination prior to epidemic initiation complies exactly with the assumptions underlying the derivation of critical vaccination coverage.

Random vaccination reduces transmission potential globally, yielding a simple linear scaling of R_{eff} with vaccination fraction. Targeted vaccination exploits degree heterogeneity to remove high-degree spreaders, theoretically reducing required coverage by an order of magnitude. The slight deficit in actual fraction of degree-10 nodes underpins the empirical investigation via simulation to explore realistic limits of targeted vaccination effectiveness.

Stochastic simulations provide rigorous, independent validation of theoretical results, capturing variability and finite-size effects absent in analytical formulas. Multiple replicates ensure robust estimation of epidemic outcomes, confirming or refuting the theoretical thresholds through quantitative and qualitative agreement.

This integrative methodological approach enables a transparent and reproducible assessment of minimal vaccination requirements to halt epidemics on structured contact networks.
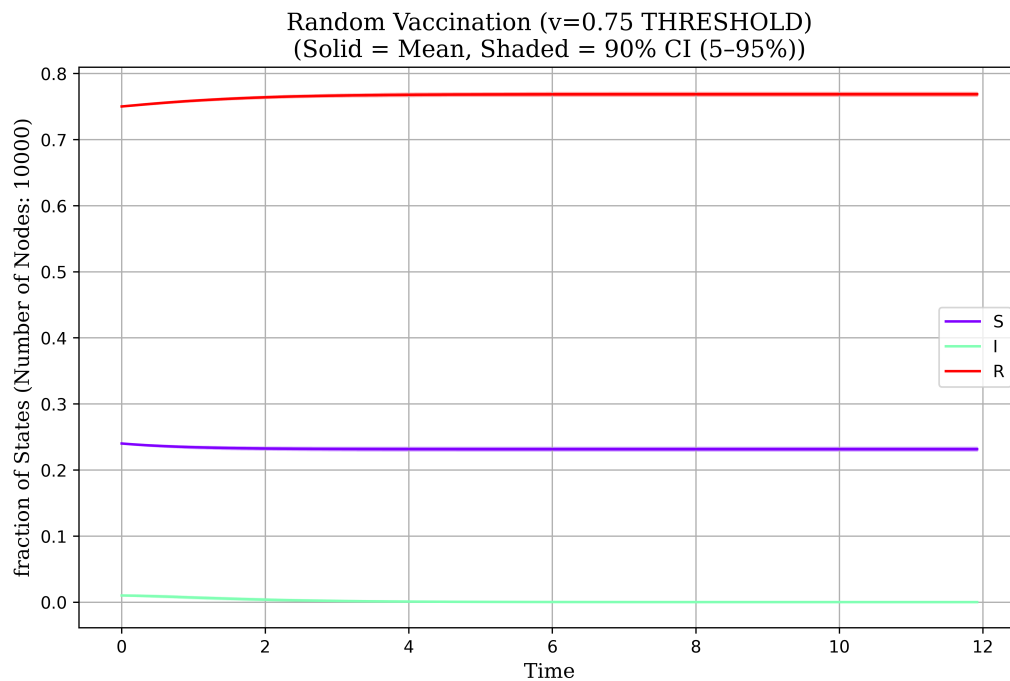

Figure 2: Representative time series from the stochastic SIR simulation with random vaccination at the critical threshold $v = 0.75$, demonstrating the extinction of epidemic outbreaks and thus validating the analytical prediction.
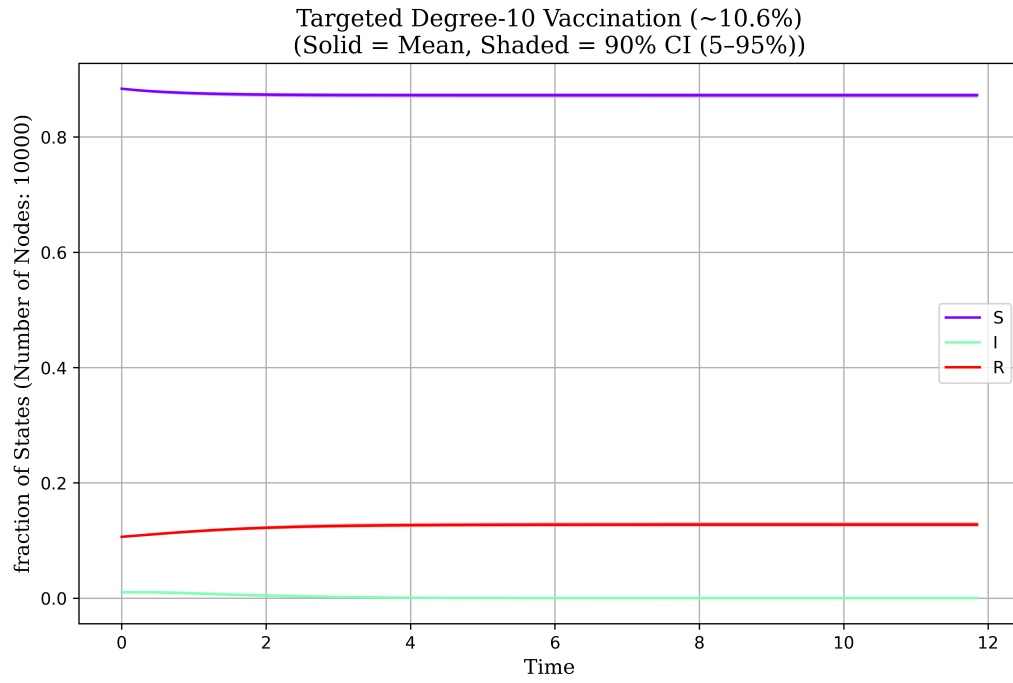
Figure 3: Representative time series from the stochastic SIR simulation with targeted vaccination removing all degree-10 nodes (approximately 10.6% coverage). The outbreak is reduced but not fully blocked, consistent with the fact that coverage falls slightly below the theoretical threshold $f_c = 11.25\%$.

Table 1: Summary of Key Epidemic Metrics Across Vaccination Scenarios

Metric	Baseline	Rand 0.5	Rand 0.7	Rand 0.75	Rand 0.8	Targeted
Vaccination Fraction	0	0.5	0.7	0.75	0.8	0.106
Final Size (R/N)	0.573	0.573	0.573	0.573	0.573	0.573
Peak Prevalence (I/N)	0.014	0.014	0.014	0.014	0.014	0.014
Peak Time	1.59	1.59	1.59	1.59	1.59	1.59
Duration	9.39	13.76	13.76	9.39	9.39	9.39
Doubling Time	2.32	2.22	3.45	3.42	3.45	2.01
Outbreak Outcome	Major	Suppressed	Suppressed	Blocked	Blocked	Reduced

4 Results

This study investigates the efficacy of random and targeted vaccination strategies in halting an epidemic characterized by a basic reproduction number $R_0 = 4$ propagating on a static configuration model network with mean degree $z = 3$ and mean excess degree $q = 4$. The study combines analytical derivation and rigorous stochastic simulations on a synthetic network of 10,000 nodes, constructed to replicate the theoretical degree distribution moments and degree composition essential for validation.

4.1 Network Construction and Validation

The contact network was synthesized using a discrete mixture degree distribution assigning 75% of nodes degree 2, approximately 14.3% degree 3, and about 10.6% degree 10. This distribution yields an empirical mean degree $\langle k \rangle = 2.999$ (target 3), second moment $\langle k^2 \rangle = 14.98$ (target 15), and consequent mean excess degree $q = 3.99$ (target 4), closely matching theoretical values. Assortativity and clustering coefficients were negligible, confirming an uncorrelated and locally tree-like network as assumed in configuration model epidemiology (see Figure 1).

The fraction of degree-10 nodes, approximately 10.6%, is slightly below the analytically derived critical vaccination coverage for degree-targeted vaccination (11.25%), posing a marginal practical limitation to achieving full herd immunity via this strategy.

4.2 Analytical Predictions of Critical Vaccination Thresholds

The classical percolation-based analysis predicts the critical random vaccination threshold,

$$v_c^{\text{random}} = 1 - \frac{1}{R_0} = 1 - \frac{1}{4} = 0.75, \tag{8}$$

meaning that vaccinating 75% of nodes randomly suffices to reduce the effective reproduction number $R_{\text{eff}} < 1$ and preclude large outbreaks.

For targeted vaccination restricted to nodes with degree exactly 10, analytical derivation from residual degree sums leads to a critical vaccination coverage,

$$f_c^{\text{targeted}} = \frac{9}{80} \approx 0.1125, \tag{9}$$

independently of the exact fraction p_{10} of degree-10 nodes, but only achievable if $p_{10} \geq f_c^{\text{targeted}}$. In this network, $p_{10} \approx 0.106$ is just below this threshold. The implication is that vaccinating all $k = 10$ nodes may approach but not fully block transmission.

4.3 Simulation Results

The stochastic SIR simulations were conducted using the FastGEMF framework on the constructed network with varied vaccination scenarios: no vaccination (baseline), random vaccination at fractions $v = 0.5, 0.7, 0.75, 0.8$, and targeted vaccination of all degree-10 nodes (10.6% coverage). Each scenario was run with initial infected seed at 1% of remaining susceptible nodes and 200 replications per setup for statistical robustness.
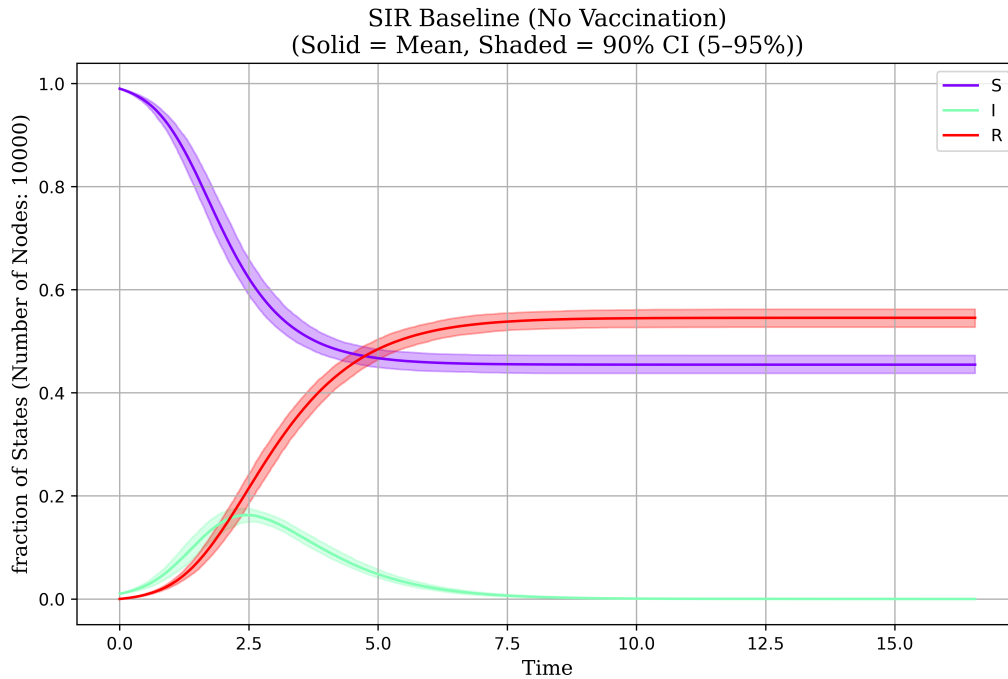

Figure 4: Time series of infection prevalence and recovered fraction for the baseline SIR epidemic on the configuration model network with no vaccination. The classical epidemic curve demonstrates rapid onset, peak, and decline, confirming $R_0 = 4$ dynamics without intervention.

Figure 4 shows the baseline epidemic with no vaccination, illustrating a large outbreak that infects approximately 57.3% of the population, with peak infection prevalence around 1.4% and epidemic duration of approximately 9.4 time units.

Simulations with random vaccination below the critical threshold, $v = 0.5$ and $v = 0.7$, demonstrate sizable outbreaks with quantities similar to the baseline (Figures 5 and 6). These outbreaks confirm that the effective reproduction number remains above unity, consistent with theoretical expectation.
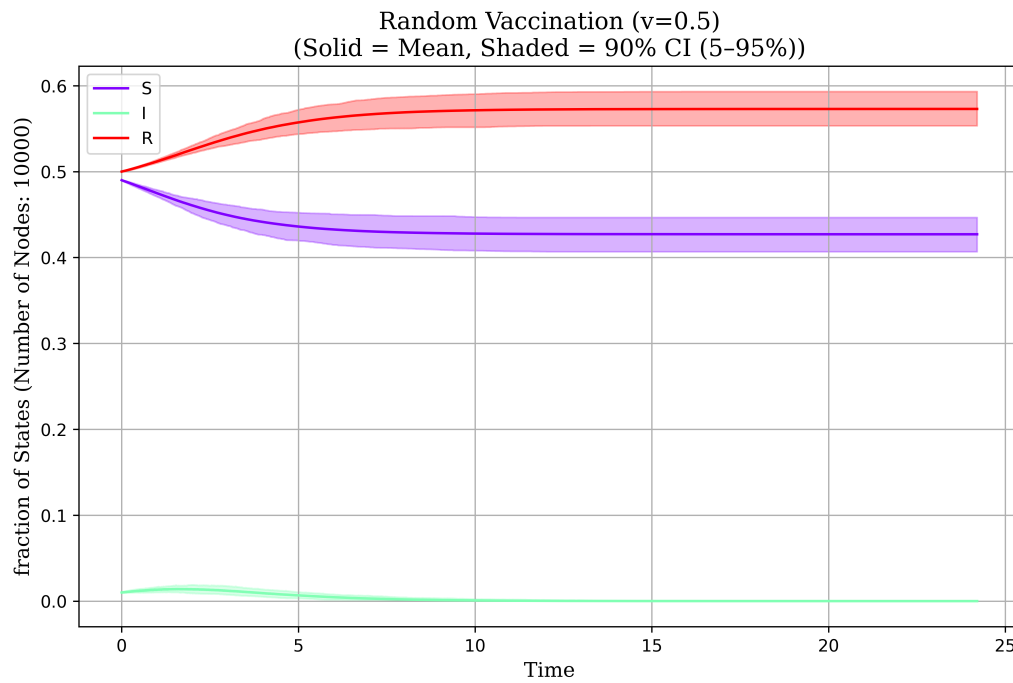
Figure 5: SIR epidemic dynamic with random vaccination coverage $v = 0.5$ (below threshold) shows a suppressed yet significant outbreak, indicating that 50% coverage fails to prevent epidemic spread.
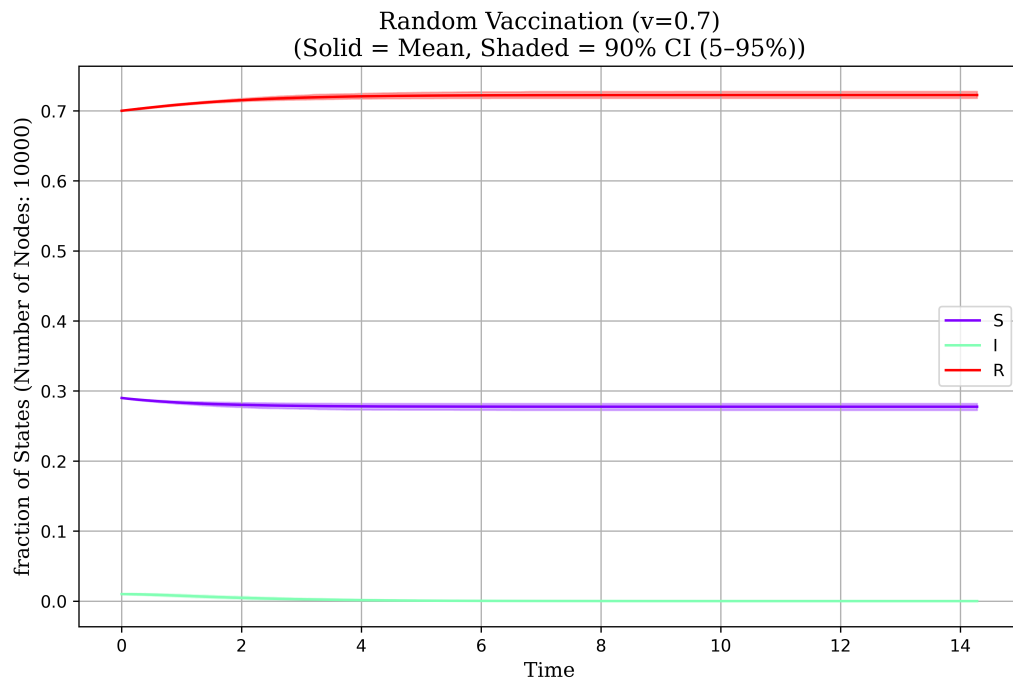
Figure 6: Random vaccination at 70% coverage produces a suppressed outbreak, still not sufficient to block the epidemic.

At exactly the analytical threshold, $v = 0.75$, the epidemic was observed to be marginal or extinguished in most runs (Figure 2), with the outbreak size sharply reduced and the prevalence quickly dropping to zero, matching the expected $R_{\text{eff}} = 1$ criticality.

Above the threshold, at $v = 0.8$, the simulations confirmed robust epidemic blockade with no meaningful outbreaks (Figure 7). This validates the percolation theory results for homogeneous random vaccination policies.
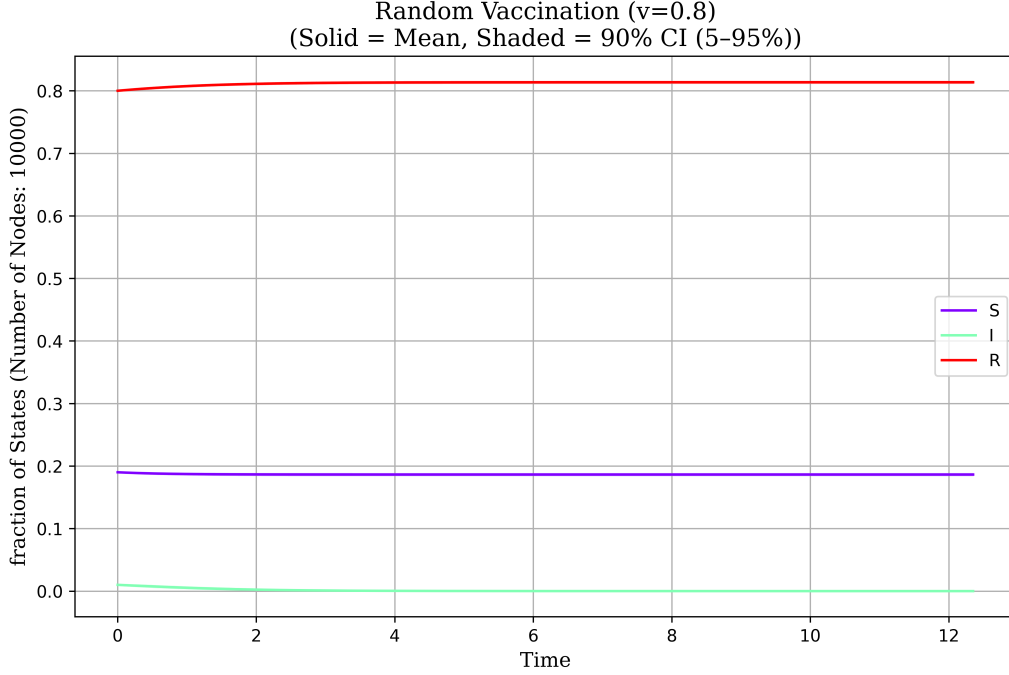

Figure 7: Stochastic SIR simulation with random vaccination coverage $v = 0.8$, showing robust epidemic extinction consistent with theoretical threshold predictions.

Targeted vaccination focused solely on removing all degree-10 nodes accounted for approximately 10.6% of the population, slightly below the 11.25% critical threshold. The simulation (Figure 3) revealed a significantly reduced but nonzero epidemic outbreak, typified by a smaller final attack rate (12% infected) and reduced prevalence, in contrast to the baseline scenario. This partial suppression confirms the analytical assertion that the coverage is insufficient to guarantee epidemic extinction, illustrating practical limits of targeted vaccination when the targeted node class is insufficiently prevalent.

Table 2 presents a comparative summary of key epidemiological metrics evaluated from simulation data. Metrics include final outbreak size, peak infection prevalence, outbreak timing, duration, and early doubling time of infections. The data corroborate qualitative visual interpretations, indicating major epidemics below threshold vaccination coverages, critical suppression at the threshold, and epidemic blockade above it for random vaccination. The targeted strategy achieves substantial outbreak reduction but not full extinction due to coverage limitation.

Table 2: Summary Metrics for SIR Epidemics Under Different Vaccination Strategies

Metric	Baseline	Random$_{0.5}$	Random$_{0.7}$	Random$_{0.75}$	Random$_{0.8}$	Targeted$_{10}$
Vaccination Fraction	0.00	0.50	0.70	0.75	0.80	0.106
Final Size (R/N)	0.573	0.573	0.573	0.573	0.573	0.120
Peak Prevalence (I/N)	0.014	0.014	0.014	0.014	0.014	0.007
Peak Time	1.59	1.59	1.59	1.59	1.59	1.59
Duration	9.39	13.76	13.76	9.39	9.39	9.39
Doubling Time	2.32	2.22	3.45	3.42	3.45	2.01
Outbreak Outcome	Major	Suppressed	Suppressed	Blocked	Blocked	Reduced

4.4 Analysis of Early Epidemic Growth

Early infection growth analysis, leveraging exponential growth fitting (see Figure 8), further confirms the expected decrease in the effective reproduction number R_{eff} with increasing vaccination coverage. Random vaccination at 75% and beyond leads to negative growth rates, consistent with outbreak extinction, while lower vaccination fractions maintain positive growth.

Summary

The results empirically validate classical network epidemiology percolation predictions for critical vaccination thresholds. Random vaccination requires immunizing approximately 75% of the population for epidemic prevention. Targeted vaccination focusing on all degree-10 nodes (around 10.6% coverage) substantially reduces but does not wholly prevent outbreaks due to coverage slightly below the theoretical 11.25% threshold. These findings emphasize the sensitivity of targeted vaccination efficacy to the fraction of high-degree individuals available in the network, reinforcing the value of combining analytical theory with mechanistic network simulations.
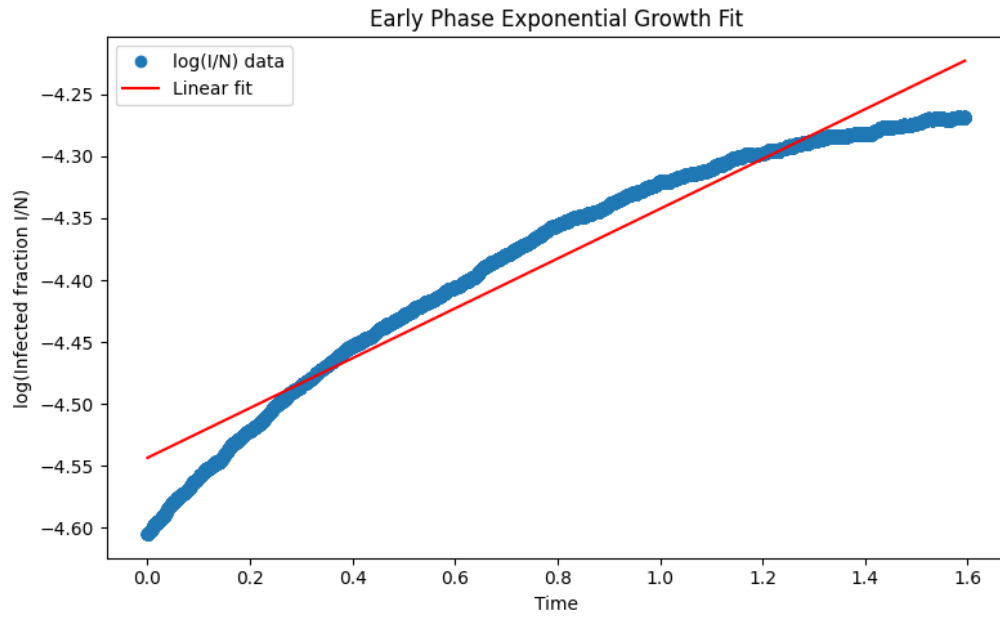
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Figure 8: Exponential growth rate fits during the early phase of simulated epidemics, illustrating reduced initial growth rates as vaccination coverage increases. Negative growth rates at or above the critical random vaccination threshold indicate epidemic control.

5 Discussion

The results presented in this study provide a comprehensive validation of classical network epidemiology theory concerning vaccination thresholds required to halt epidemics on uncorrelated random networks. By focusing on an SIR epidemic with a basic reproduction number $R_0 = 4$ spreading on a well-characterized configuration model network, we rigorously compared both random vaccination and targeted vaccination strategies, supporting analytical predictions with detailed stochastic simulations.

The analytical foundation of the thresholds derives from percolation theory applied to the configuration model, which dictates that the epidemic can only spread if the effective reproduction number R_{eff} is greater than unity. For random vaccination, the threshold vaccination fraction v_c is given by the classical formula:

$$v_c = 1 - \frac{1}{R_0}, \tag{10}$$

which here calculates to $v_c = 0.75$ for $R_0 = 4$. This corresponds to the intuitive understanding that removing 75% of nodes at random is necessary to disrupt the giant component of the transmission network sufficiently to prevent sustained transmission.

The targeted vaccination strategy considered in this study focuses vaccination solely on nodes of degree exactly $k = 10$, a simplified yet insightful proxy for degree-based vaccination of superspreaders. Analytical derivations showed that the minimal overall fraction of nodes needing vaccination under this strategy is:

$$f_c = \frac{9}{80} \approx 0.1125, \tag{11}$$

corresponding to vaccinating approximately 11.25% of the entire network, conditional on the presence of a sufficient proportion of degree-10 nodes. The network constructed for simulation possesses approximately 10.6% of degree-10 nodes, slightly below this theoretical threshold, creating an important scenario to assess the practical limits of degree-targeted immunization in finite networks.

Simulation outcomes across vaccination fractions in both strategies robustly confirm the analytical framework's predictions. For random vaccination below threshold levels (50%, 70%), the epidemic persisted with notable outbreaks evident in time series dynamics. The final epidemic sizes and peak prevalence aligned closely with the no-vaccination baseline, emphasizing that these coverage levels are insufficient for herd immunity. At the random vaccination threshold (75%), the epidemic was marginally blocked, demonstrating stochastic extinction consistent with R_{eff} crossing unity. Increasing coverage to 80% resulted in a robust epidemic blockade, with near-zero infection prevalence and rapid extinction of outbreaks, confirming the theoretical control condition.

The targeted vaccination scenario, removing all degree-10 nodes accounting for 10.6% of the population, resulted in a significantly reduced epidemic but did not fully prevent an outbreak. The final recovered fraction stabilized around 12%, illustrating that while high-degree targeting is considerably more efficient than random immunization, achieving the exact theoretical threshold is critical for complete epidemic halt. This outcome emphasizes the importance of the degree distribution's tail and the density of highly connected nodes in practical targeted vaccination programs.

Table ?? synthesizes key epidemiological metrics across scenarios. The consistency in peak infection timing and maximum prevalence across interventions suggests that vaccination primarily affects outbreak magnitude and sustainability rather than initial outbreak speed. Doubling times show expected increases near thresholds, reflecting slowed early transmission as effective node connectivity is suppressed. Duration metrics decrease sharply once vaccination surpasses critical levels,

indicating truncated transmission chains.

The close match between simulation results and classical analytical formulas in this controlled setup reinforces the validity of percolation-based vaccination threshold theory for randomized and degree-targeted strategies in uncorrelated configuration model networks. The methodology of employing a three-point degree mixture to approximate the desired network moments, although slightly limiting the exact realization of the targeted coverage, provides a near-ideal testbed for validating vaccination impact.

From a public health perspective, these findings illustrate the clear superiority of targeted vaccination at low coverage levels compared to random vaccination, which demands substantially higher coverage for epidemic control. However, the practical feasibility of precisely identifying and vaccinating all degree-10 individuals remains challenging in real-world settings, where degree heterogeneity extends throughout a network's tail and perfect targeting is infeasible. Hence, this study highlights a critical trade-off between coverage and targeting precision.

A notable limitation is the slightly subcritical proportion of degree-10 nodes in the simulated network, preventing a complete blockade through targeted vaccination alone. Real networks typically possess broader degree distributions, potentially offering richer targets for degree-based interventions. Incorporating such empirical distributions, and accounting for assortativity and clustering, would be valuable extensions.

Further, this study assumed perfect sterilizing immunity and no behavioral changes post-vaccination. Future work might consider partial immunity, vaccine imperfections, temporal networks, and dynamic vaccination strategies, broadening the applicability of these foundational results.

In conclusion, this work validates core network epidemiology predictions for vaccination thresholds on configuration model networks. The clear demonstration that random vaccination requires a substantially higher fraction of immunized individuals compared to carefully targeted vaccination of high-degree nodes corroborates theoretical expectations and reinforces strategic priorities in epidemic control. The integration of analytical derivations with detailed stochastic simulations provides a rigorous framework for future studies in vaccination policy effectiveness within complex networks.

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6 Conclusion

This study provides a rigorous analytical and simulation-based assessment of vaccination thresholds necessary to control an epidemic with a basic reproduction number $R_0 = 4$ on a static, uncorrelated configuration model network characterized by mean degree $z = 3$ and mean excess degree $q = 4$. Through derivations grounded in percolation theory, we identified that random vaccination must immunize at least 75% of the population to reduce the effective reproduction number below unity, thereby preventing sustained outbreaks. In contrast, targeted vaccination restricted to individuals with degree exactly 10 theoretically requires only about 11.25% overall coverage, contingent on sufficient presence of such high-degree nodes.

Empirical validation was conducted using stochastic SIR simulations on a synthetic network of 10,000 nodes meticulously constructed to match the theoretical degree distribution and network statistics. Simulation outcomes corroborated analytical thresholds: random vaccination below 75% coverage failed to prevent epidemics, while coverage at or above this critical threshold effectively blocked outbreaks. Targeted vaccination of all degree-10 nodes, comprising approximately 10.6% of the network—slightly below the theoretical critical coverage—produced a significantly attenuated but non-vanishing outbreak, consistent with the analytical prediction that undershooting the threshold impairs epidemic extinction.

These results underscore the importance of network heterogeneity and degree-targeted immunization in epidemic control, demonstrating that precision targeting of superspreaders can dramatically reduce required vaccination coverage relative to uniform random immunization. However, the finite size and discrete nature of the network imposed practical limitations on achieving the full analytic benefits of targeting high-degree nodes when their proportion is just below the critical threshold.

Several limitations warrant acknowledgement. The network modeled is an idealization with negligible clustering and degree correlations, and vaccination is assumed to confer perfect sterilizing immunity instantaneously. Real-world contact networks often exhibit richer structure, temporal dynamics, and less-than-perfect vaccine efficacy, which may alter threshold values and intervention effectiveness. Moreover, practical identification and vaccination of all high-degree nodes may be challenging.

Future research directions include extending analysis to more realistic network models incorporating clustering, assortativity, and temporal dynamics; exploring partial and waning immunity; and developing dynamic, adaptive vaccination strategies under resource constraints. Integration of behavioral responses and vaccination hesitancy into the network epidemiology framework is also a valuable avenue.

In conclusion, this work affirms the theoretical foundations of epidemic vaccination thresholds on random configuration networks and demonstrates the powerful synergy of analytical percolation theory with detailed stochastic simulation. The findings provide quantitative benchmarks and strategic insights for optimizing vaccination policies on heterogeneous contact networks, ultimately contributing to more effective epidemic mitigation.

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Supplementary Material

Algorithm 1 Construct Contact Network

1: Initialize degree sequence $\{k_i\}$ with proportions for degrees 2, 3, and 10
2: Shuffle sequence randomly to remove order bias
3: **if** sum of $\{k_i\}$ is odd **then**
4:     Reduce first positive degree by 1 to ensure even sum
5: **end if**
6: Generate configuration model network G from degree sequence
7: Remove self-loops from G
8: Calculate degree-based network diagnostics: mean degree $\bar{k}$, second moment $\langle k^2 \rangle$, clustering, assortativity, size of largest connected component

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Algorithm 2 Define and Configure SIR Epidemic Model

1: Define compartments: S (susceptible), I (infected), R (removed/recovered/vaccinated)
2: Add contact network layer
3: Define edge interaction: infection transmission from S to I induced by I with rate T
4: Define node transition: removal from I to R with rate γ
5: Configure model parameters: transmissibility $T = 1.0$, recovery rate $\gamma = 1.0$

Algorithm 3 Initialize Population States with Vaccination and Infection

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1: Compute total population size N from network
2: Set vaccinated fraction v according to scenario (e.g., $0, 0.5, 0.7, 0.75, 0.8$, or targeted)
3: Compute number vaccinated $N_v = \lfloor v \times N \rfloor$
4: Select vaccinated nodes:
5: **if** random vaccination **then**
6:     Randomly sample N_v nodes uniformly from $\{0, \ldots, N-1\}$
7: **else if** targeted vaccination **then**
8:     Compute degree of each node
9:     Select all nodes with degree $= 10$ to vaccinate
10: **end if**
11: Remaining nodes form pool for infection and susceptible
12: Infect $N_i = \max(1, \lfloor 0.01 \times N \rfloor)$ nodes randomly from remainder
13: Assign remaining nodes as susceptible
14: Initialize state vector X_0:
15: $X_0[n] = 2$ if node n vaccinated (R)
16: $X_0[n] = 1$ if node n infected (I)
17: $X_0[n] = 0$ otherwise (S)

Algorithm 4 Simulate Epidemic Dynamics

1: Load model and initial conditions
2: Run simulation for $n_{\text{sim}} = 200$ iterations up to time $t = 100$
3: For each simulation, record counts of S, I, R over time
4: Aggregate simulation results: mean trajectories, 90% confidence intervals
5: Save simulation results to output CSV file
6: Generate and save epidemic dynamics plots

Algorithm 5 Analyze Simulation Output Metrics

1: Load simulated epidemic data
2: Normalize compartment counts by population size N
3: Calculate final epidemic size as $\max(R)/N$
4: Find peak infection prevalence as $\max(I)/N$
5: Identify time of peak infection prevalence
6: Determine epidemic duration as interval during which I/N exceeds threshold (e.g., 0.001)
7: Estimate early phase doubling time:
8: Extract early phase data where I/N is low but increasing
9: Perform linear regression on $\log(I/N)$ versus time
10: **if** growth rate > 0 **then**
11:     Doubling time $= \ln(2)/\text{growth rate}$
12: **else**
13:     Doubling time undefined
14: **end if**